(presumably α_{1B} and α_{1L} subtypes), both of which are involved in the noradrenaline-induced contraction.

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MINOR ROLE FOR DIRECT ADRENOCEPTOR-MEDIATED CALCIUM ENTRY IN RAT MESENTERIC RESISTANCE ARTERIES

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Ca^{2+}-entry into vascular smooth muscle cells occurs through potential-dependent, dihydropyridine-sensitive calcium channels, and possibly also through calcium channels which are activated by receptor activation, independent of membrane potential (ROCs). We have investigated the possible functional role of adrenoceptor-mediated ROCs in rat mesenteric small arteries from Wistar rats. $[Ca^{2+}]_i$ determined by fura-2 fluorescence and force were measured simultaneously using a myograph. Depolarization with high-potassium solution (K-PSS) elevated $[Ca^{2+}]_i$ and induced contraction. Further addition of 10 µM noradrenaline (NA) did not elevate $[Ca^{2+}]_i$ further, but enhanced tone, thus confirming that agonist activation causes calcium-sensitization. When this protocol was repeated in the presence of calcium channel inhibitors (felodipine (1 nM) or D-600 (1 µM)), K-PSS induced neither maintained tone nor maintained elevation of $[Ca^{2+}]_i$, but the further addition of NA still caused a rise in tone and $[Ca^{2+}]_i$, to about half the previous levels. Repetition of the protocol in the presence both of calcium channel inhibitors and also drugs which deplete intracellular calcium stores (ryanodine (10 µM) or thapsigargin (1 µM)), showed that the NA-induced rise in $[Ca^{2+}]_i$ was inhibited, but a small (10-20% of control) rise in tone remained. The experiments suggest that ROCs do not contribute substantially to control of $[Ca^{2+}]_i$, and that effects of NA on tone which are independent of membrane potential are due primarily to calcium-sensitization but also to release of Ca^{2+} from intracellular stores.

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MODE OF EXISTENCE OF 74 KDA PROTEIN, ONE OF THE GELSOLIN FAMILY IN DEVELOPMENTAL STAGE OF VASCULAR SMOOTH MUSCLE

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The 74 KDa protein of the gelsolin family is distributed in non-muscle cells with exocytic function, but not in normal adult smooth muscle cells. In primary culture of smooth muscle from bovine carotid artery, we found that this protein was gradually expressed, arriving at maximum in confluent state by immunoblotting method. On the other hand, in the immunofluorescent method of the cultured cells 74 Kda protein was not expressed in all the cells with deformed globular feature on the 1st day without serum, but smooth muscle type of alpha-actin and smooth muscle myosin were clearly expressed. On 4th day after addition of serum, 74 KDa protein was expressed at the surface membrane of all the globular cells of which numbers were slightly increased by slow cell division. At time bipolar cells appeared underneath the globular cells. These cells had no 74 Kda protein and alpha-actin. Once bipolar cells appeared, their growth rate was very fast and their numbers increased rapidly. When cells were proceeding to confluent state, accompanied by hills and valleys structure, globular cells were gathered, and formed tight round structure on the top of the hills. At that time alpha-actin began to be expressed in abundant bipolar cells.

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CORONARY VASODILATOR RESPONSE TO BRADYKININ: ROLE OF CYTOCHROME P450 AND K^+ CONDUCTANCE

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The rat coronary vasodilator response to bradykinin (BK) is reportedly independent of NO and cyclooxygenase and lipoxygenase products of arachidonic acid (AA). We investigated the role of cytochrome P450 (P450) in the coronary vasodilator response to BK using the Langendorff heart preparation which was preconstricted with nitroarginine (50 µM) to eliminate NO. As reported, neither nitroarginine nor indomethacin (2.8 µM) affected the coronary vasodilator

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